

by the detection of transmural fat were 94% and 90% respectively. Defined by wall motion abnormalities, the sensitivity and specificity were 88% and 100%. **Conclusion:** MRI seems to be a sensitive and specific method for the diagnosis of ARVD by assessing in the same region tissue abnormalities and regional dysfunction of the right ventricle.

1091-95 Is Inducible Sustained Monomorphic Ventricular Tachycardia a Reliable Endpoint for Treatment Decisions in Patients with Asymptomatic Nonsustained Ventricular Tachycardia and Ejection Fraction ≤ 40 Percent?

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Recent clinical trials have utilized inducible sustained monomorphic ventricular tachycardia (SMVT) in pts with nonsustained ventricular tachycardia (NSVT) as an endpoint for treatment decision making. We prospectively evaluated the reproducibility of inducible SMVT on 2 consecutive days in 43 pts with asymptomatic NSVT (mean consecutive beats 18, mean rate 184 beats/min) and reduced ejection fraction (mean 26%). Reproducible SMVT was defined as inducible on 2 consecutive days using the same stimulation protocol; SMVT induced on 1 day only was classified as nonreproducible. SMVT was inducible in 21/43 (49%) pts on either day alone, but was reproducible on 2 consecutive days in only 8/21 (38%) with inducible SMVT. Nonreproducible SMVT was observed in 13/21 (62%). The remaining 22/43 pts (51%) were noninducible on either day. The overall concordance of stimulation on 2 consecutive days was 70%. Baseline variables including age, gender, heart disease, prior myocardial infarction, heart failure class, ejection fraction and duration or rate of NSVT were not predictive of either inducibility or reproducibility. **Conclusions:** Inducible SMVT is reproducible on 2 consecutive days in a minority of pts (38%) with asymptomatic NSVT and reduced ejection fraction. Limited reproducibility suggests inducible SMVT may not be a reliable endpoint for treatment decisions in these pts.

1091-96 Cerebral Dysautoregulation and Vasoneural Uncoupling During Ventricular Tachycardia

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Optimal therapy of ventricular tachycardia (VT) with implanted cardioverter-defibrillator (ICD) depends, in part, on intact cerebrovascular autoregulation and vasoneural coupling. Therefore, we examined the nature and incidence of dysautoregulation (DA) and uncoupling in 15 conscious patients, ages 66 ± 6 years, with inducible pace-terminable VT in supine (S) and head-up tilt (HUT) positions. Cerebral perfusion was quantified by changes in middle cerebral artery blood flow velocity (CBFV) measured by transcranial Doppler ultrasound. Vasoneural coupling was assessed by regional cerebral venous oxygen saturation (rCVOS) using transcranial near-infrared spectroscopy. VT was induced using the noninvasive stimulation and terminated by ATP or shock via the implanted ICD. **Results:**

| | Normal n = 6 | DA n = 5 | Uncoupling n = 4 |
|----------------------|-----------------|---------------|---------------------|
| CBFV (HUT) | | | |
| % supine baseline | 112 \pm 21 | 65 \pm 9* | 101 \pm 11 |
| rCVOS | | | |
| supine | 95 \pm 3 | 94 \pm 3 | 93 \pm 5 |
| upright | 91 \pm 4 | 87 \pm 6 | 78 \pm 6* |
| VT cycle length (ms) | | | |
| supine | 280 \pm 50 | 300 \pm 100 | 300 \pm 50 |
| upright | 300 \pm 70 | 280 \pm 90 | 320 \pm 50 |
| VT duration (s) | | | |
| supine | 14 \pm 5 | 15 \pm 6 | 17 \pm 8 |
| upright | 18 \pm 2 | 15 \pm 7 | 29 \pm 21 |
| Symptoms (HUT) | none | none | syncope |

*P < 0.01

Conclusion: HUT identified patients with cerebral dysautoregulation (decrease in CBFV with intact rCVOS) and no neurologic symptoms and vasoneural uncoupling (decrease in rCVOS despite adequate CBFV) with syncope. Upright tolerance to VT depends on cerebral vasoneural coupling independent of VT cycle length and duration.

1091-97 Delay of Local Depolarisation/Repolarisation Processes After Acute Myocardial Infarction and Impairment of Sympathetic Innervation

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Background: Regional cardiac denervation was defined scintigraphically by mismatch between ^{201}Tl and ^{123}I -MIBG defect. The goal of this study was to determine whether there is a correlation between mismatch size and electrophysiologic abnormalities in post-MI patients. **Patients:** We studied 67 consecutive patients who survived the coronary-care-unit phase of acute myocardial infarction. **Methods:** ^{201}Tl and ^{123}I -MIBG images was measured in the 2nd week after infarction, using a threshold of 50% of peak counts based on a semiquantitative polar map approach. Mismatch size was defined as difference between the defect sizes of ^{201}Tl and ^{123}I -MIBG images. QT interval, QTc and spatial QT-dispersion were derived from the resting ECG. The frequency of ventricular premature complexes (VPCs), couplets, salvos as well as heart rate variability measures were quantified in the Holter ECG. Terminal LAS, terminal RMS and QRS width were measured in the signal averaging ECG. **Results:** The $^{201}\text{Tl}/^{123}\text{I}$ -MIBG mismatch size was positively correlated with QTc ($p < 0.01$) and negatively correlated with the terminal RMS signal ($p < 0.01$). There was no significant correlation of the mismatch size with the number of VPCs, couplets and salvos, heart rate variability measures, terminal LAS, QRS width, non-correlated QT and spatial QT dispersion. **Conclusion:** Lower terminal RMS signals longer QTc are correlated with the size of ^{201}Tl and ^{123}I -MIBG mismatch. These findings suggest relatively slow depolarisation and repolarisation processes within the denervated but viable myocardium and could contribute to the genesis of late potentials.

1091-98 Noninvasive Risk Stratification in Patients with Congestive Heart Failure: Comparison of Traditional Risk Markers and T Wave Alternans

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Patients with chronic congestive heart failure (CHF) are at high risk for malignant ventricular arrhythmias and death. The following tests were performed for risk stratification in 70 patients with symptomatic CHF (left ventricular ejection fraction (LVEF) $28 \pm 7\%$, age 57 ± 10 years, 56 pts with ischemic CHF, 14 pts non-ischemic CHF): echocardiography or contrast ventriculography for determination of LVEF, 24 h Holter ECG for quantitation of arrhythmias and analysis of heart rate variability (HRV), signal averaged ECG for evaluation of late potentials, detection of T wave alternans (TWA) from the surface ECG (spectral method during rest and exercise, $n = 52$ pts). QT dispersion was determined from a 12-lead surface ECG. During follow-up of 12 ± 4 months, 13 pts had major clinical events, prospectively defined as death ($n = 3$), need for cardiac transplantation ($n = 4$) and occurrence of VF or sustained VT ($n = 6$). Patient groups with and without clinical events were compared by means of an unpaired t-test or chi-square test/Fisher's exact test (see table for results).

| | Pts with events (n = 13) | Pts without events (n = 57) | p-value |
|----------------------|-----------------------------|--------------------------------|---------|
| LVEF \pm SD (%) | 27 \pm 5 | 30 \pm 8 | < 0.01 |
| HRV (SDNN) | 80 \pm 37 | 87 \pm 35 | ns |
| HRV (rMSSD) | 32 \pm 23 | 25 \pm 13 | ns |
| PVCs/day | 705 | 1140 | ns |
| VT-episodes/day | 3.0 \pm 9.0 | 2.6 \pm 10.0 | ns |
| Late potentials + | 7/13 (58%) | 30/51 (58%) | ns |
| QT Dispersion (msec) | 66 \pm 30 | 64 \pm 24 | ns |
| TWA + or ++ | 9/9 (100%) | 26/43 (60%) | < 0.02 |
| TWA ++ | 6/9 (67%) | 11/43 (26%) | < 0.03 |

Conclusion: All diagnostic tests performed for risk stratification exhibit severe abnormalities in this population. TWA appears to be a strong risk marker in patients with CHF.

1091-99 Gender Effects of Quinidine on QT Interval and QT Dispersion

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Recent work has shown that increased QT dispersion (QTd) is associated with quinidine (Q) induced torsades de pointes (TdP). Also, women are at